which will be marketed by Rentokil Initial as an insecticide (PT18).

Given the above, and for the following reasons, it is not considered necessary to perform further testing:
The Biocidal Products Directive (98/8/EC, "the Directive") requires long-term testing in rodents as part of the suite of toxicology tests in order to assess the possible adverse consequences of chronic exposure (i.e. chronic toxicity and carcinogenicity) to the biocidal active substance. The Directive states in Article 8 (5) that "information which is not necessary owing to the nature of the biocidal product or its proposed uses need not be supplied. The same applies where it is not scientifically necessary or technically possible to supply the information. In such cases, a justification acceptable to the competent authority must be submitted..." A more detailed waiving concept is given in the TNsG on data requirements. In addition, the TNsG gives the strong recommendation "to minimise testing on vertebrate animals or to avoid unnecessary suffering of experimental animals the data should not be generated".

Behind this background, the waiver concept outlined in the TNsG on data requirements is considered applicable for silicon dioxide with regard to the teratogenicity studies and therefore a scientific justification for waiving these studies are presented below.

- It is not scientifically necessary on the basis of low exposure to silicon dioxide during its normal use as a biocide.

Exposure to amorphous silicon dioxide when used as an insecticide is inconsequential because of the ubiquity of forms of silicon dioxide in the environment. Silicon, in the form of silicon dioxide and silicates (salts of the various silicic acids), occurs abundantly in nature, comprising about 25% of the earth's crust. Silicon dioxide and silicates are present in practically all plants and animals and in natural waters. Between 10 and 200 mg silicon dioxide is present in 100g dry weight of normal human tissue. The lungs and lymph nodes of older adults may have levels several times this amount. Silicon dioxide is an approved food additive, assigned the E number E551, and is used as an anti-caking agent. Silicon dioxide has been given an acceptable daily intake of "not limited". In addition, silicon dioxide is approved for use in plastic material coming into contact with food, without hazard to public health. Synthetic amorphous silicas are widely used in industry (for example as absorbents, dessicants and fillers) and in synthetic fabrics, plastics, lacquers, vinyl coatings, varnish, paper, pharmaceuticals, adhesives, foods, floor waxed, paints rubber, and inks. Estimates indicate that 4,400,000 people are exposed to amorphous silicas in their work environments. The risk assessment for human exposure to silicon dioxide, when applying the representative product RID Insect Powder, estimates exposure to be 0.0043 mg silicon dioxide/kg/day *. To put this exposure into context, and notwithstanding the information given above, the silicon dioxide content of raw potato is reported to be 10.1 mg/kg, and one litre of beer contains 131 mg.
* Refer to Document IIIA, section 2.10 for details of human risk assessment for silicon dioxide.

- In addition to the above, the potential for exposure to silicon dioxide when it is manufactured for use as an insecticide is minimal. Silicon dioxide is manufactured in a completely enclosed system, as is the manufacture of the insecticide product based on silicon dioxide. This means there is no exposure to workers, bystanders or the environment during manufacture. It is estimated that [redacted] of silicon dioxide will be manufactured each year for use as a biocide. This amount of silicon dioxide is tiny in comparison to the other non-biocidal uses of silicon dioxide. For example, amorphous silicon dioxide is the main component of glass and in 1995, 12.9 million tonnes of glass was discarded in the US alone.

  [redacted]

- Operator exposure work has been carried out in humans exposed to high concentrations of silicon dioxide. Such data has been used previously by a number of regulatory authorities to set national, international and supranational maximum exposure limits for safe working conditions, and all of these exposure limits are in general agreement. For example, the long term occupational exposure limit for silicon dioxide set in the UK is 2.4 mg/m^3 (respirable dust) (8h time weighted average). The US threshold limit value (TLV, set by the American Conference of Governmental Industrial Hygienists, ACGIH) for silicon dioxide is 2 mg/m^3 (respirable dust). In Australia, the long-term occupational exposure limit for silicon dioxide is also 2 mg/m^3 (respirable dust). The risk assessment for human exposure to silicon dioxide, when applying the representative product, RID Insect Powder shows that exposure to silicon dioxide does not exceed these agreed maximum exposure limits for safe working conditions*. As the objective of an animal test is to predict the toxicological effect in humans, then an established safe exposure level based on human data takes precedence over animal data generated for an approximation of a theoretical safe value.

*The risk assessment for human exposure to silicon dioxide shows exposure to RID Insect Powder, under normal working conditions did not exceed the recommended UK maximum exposure limit to amorphous silicon dioxide (set at 2.4 mg/m^3 for respirable dust)**.

** Refer to Document IIIA, section 2.10 for details of human risk assessment for silicon dioxide.

- There is a substantial volume of information available for amorphous silicon dioxide. The data available are in general agreement, all showing that amorphous silicon dioxide *per se* is intrinsically biologically inert.

There is a substantial volume of information available for silicon dioxide, and while there are no studies available performed to specific guidelines, which consider chronic toxicity or genotoxicity specifically, it does cover all the major biological considerations. Given the large volume of data available for silicon dioxide, only the typical findings have been summarised below with regards to the chronic toxicity and carcinogenic potential of silicon dioxide. A number of reviews have been conducted by different regulatory bodies including the EPA, and the FDA, who considered the health aspects of silicon dioxide as a food additive. EPA concluded that silicon dioxide's acute toxicity profile is characterised as moderate to low, and consequently silicon dioxide has been exempted from the requirement of a tolerance limit when applied to growing crops or agricultural commodities. FDA has classified silicon dioxide as Generally Recognised as Safe (GRAS) and has approved its use as a dietary food additive at levels of up to 2% by weight in food. The joint FAO/WHO Expert Committee evaluated a number of food additives. The anti-caking agent silicon dioxide was given an acceptable daily intake of "not limited". There are two FDA direct food ingredient regulations for silicon dioxide, plus a clearance by the US Department of Agriculture for its use in curing mixes and in animal feed premixes. In agreement with the review by the EPA, the FDA concluded that silicon dioxide appears to be biologically inert and there was no evidence available that suggests silicon dioxide is hazardous to humans.

Exposure to increasing concentrations of silicon dioxide: Effects and observations

Below is a summary of the long-term toxicity studies available for silicon dioxide. They are summarised in full under the relevant end points in Document IIIA.

Chronic, oral
Takizawa et al. orally administered 0, 0.125, 2.5 and 5% amorphous silica to $B_6C_3F_1$ mice and Fisher rats 93 weeks and 103 weeks respectively and found that repeated oral administration produced no significant treatment-related effects. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – Study summary 1 of 1).

Chronic, inhalation
Schepers exposed Wistar rats, guinea pigs and rabbits to 126 mg/m^3 amorphous silica by inhalation for a maximum of 24 months. No radiographic signs of lung disease in animals at the end of their maximal period of silicon dioxide inhalation were found. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – Study summary 1 of 2).

Choudat et al studied the health records and chest x-rays of 131 workers (male), 90 of which were the control group and 41 of which were the test group. The 41 men were exposed to 0 – 3.4 mg/m^3 respirable dust over a mean exposure period of 8 years. It was shown that the exposure to precipitated silica dust induces little respiratory impairment, which was increased by smoking. The test subject questionnaire, chest x-ray films and concentrations of arterial blood gas were used to distinguish the two groups of workers (exposed or not) None of these methods were able to discriminate. Exposure to amorphous silica dust may induce a mild small airway disease, only in comparison to a control group. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – study summary 2 of 2).

Repeated dose, inhalation
Reuzel et al. exposed Wistar rats to up to 30 mg/m^3 amorphous silica by inhalation for 90 days. It was found that amorphous silicas did not induce persistent granulomas and the adverse affects in the respiratory tract partly or completely regressed. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.4 – Study summary 1 of 2).

Johnston et al. exposed Fischer-344 rats to 50 mg/m^3 amorphous silica by inhalation for 90 days. It was found that amorphous silicon dioxide did not cause gene mutation, partly because

of its low biopersistence and that the effects of exposure were reversible as demonstrated by the post-exposure results. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.4 – Study summary 2 of 2).

Carcinogenicity
Takizawa et al. orally administered 0, 0.125, 2.5 and 5% amorphous silica to $B_6C_3F_1$ mice and Fisher rats 93 weeks and 103 weeks respectively and found that repeated oral administration produced no significant treatment-related effects. (Referenced and summarised in Document IIIA, Annex point IIA, 6.7 – Study summary 1 of 1).

Conclusion

It has been demonstrated that the low level of exposure to silicon dioxide during its use as an insecticide (PT18) indicates that it is not scientifically necessary to conduct a teratogenicity study on silicon dioxide as it will not add any useful information to the risk assessment. It has been shown in the human risk assessment that compared to exposures *via* the diet and the environment, exposure from silicon dioxide as an insecticide is insignificant. The risk assessment for human exposure to silicon dioxide, when applying the representative product RID Insect Powder shows that exposure to silicon dioxide does not exceed agreed, well established maximum exposure limits for safe working conditions with silicon dioxide and nuisance dust. The toxicological profile of silicon dioxide has been well established with a large body of data available in the public domain. The operator exposure limits that have been set for nuisance particles and dusts are also based on a large amount of available data. As shown above, data is available on the effects of exposure to amorphous silicon dioxide and this data shows that there are no lasting adverse effects. Although this data has its limitations and there are no studies available performed to specific guidelines which consider chronic toxicity or genotoxicity specifically, it is considered sufficient to address the toxicity of silicon dioxide particularly given the levels of exposure expected to silicon dioxide through other, non-biocidal uses of silicon dioxide including its use in food.

3.8.2 Fertility

Route of exposure	Test type Method guideline	Species Strain Sex No/group	Exposure Period	Doses	Critical effect	NO(A)EL Parental		NO(A)EL FI		NO(A)EL F2		Remarks	Reference
						m	f	m	f	m	F		
Not applicable	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable		Not applicable		Not applicable		See below.	Document III-A6 Section 6.8.2

It is not considered necessary to perform tests on the effects of amorphous silicon dioxide over multiple generations for the following reasons:
The Biocidal Products Directive (98/8/EC, "the Directive") requires long-term testing in rodents as part of the suite of toxicology tests in order to assess the possible adverse consequences of chronic exposure (i.e. chronic toxicity and carcinogenicity) to the biocidal active substance. The Directive states in Article 8 (5) that "information which is not necessary owing to the nature of the biocidal product or its proposed uses need not be supplied. The same applies where it is not scientifically necessary or technically possible to supply the information. In such cases, a justification acceptable to the competent authority must be submitted..." A more detailed waiving concept is given in the TNsG on data requirements. In addition, the TNsG gives the strong recommendation "to minimise testing on vertebrate animals or to avoid unnecessary suffering of experimental animals the data should not be generated".

Behind this background, the waiver concept outlined in the TNsG on data requirements is considered applicable for silicon dioxide with regard to the multi-generations study and therefore a scientific justification for waiving this study are presented below.

- It is not scientifically necessary on the basis of low exposure to silicon dioxide during its normal use as a biocide.

Exposure to amorphous silicon dioxide when used as an insecticide is inconsequential because of the ubiquity of forms of silicon dioxide in the environment. Silicon, in the form of silicon dioxide and silicates (salts of the various silicic acids), occurs abundantly in nature, comprising about 25% of the earth's crust. Silicon dioxide and silicates are present in practically all plants and animals and in natural waters. Between 10 and 200 mg silicon dioxide is present in 100g dry weight of normal human tissue. The lungs and lymph nodes of older adults may have levels several times this amount. Silicon dioxide is an approved food additive, assigned the E number E551, and is used as an anti-caking agent. Silicon dioxide has been given an acceptable daily intake of "not limited". In addition, silicon dioxide is approved for use in plastic material coming into contact with food, without hazard to public health. Synthetic amorphous silicas are widely used in industry (for example as absorbents, dessicants and fillers) and in synthetic fabrics, plastics, lacquers, vinyl coatings, varnish, paper, pharmaceuticals, adhesives, foods, floor waxed, paints rubber, and inks. Estimates indicate that 4,400,000 people are exposed to amorphous silicas in their work environments. The risk assessment for human exposure to silicon dioxide, when applying the representative product RID Insect Powder, estimates exposure to be 0.0043 mg silicon dioxide/kg/day *. To put this exposure into context, and notwithstanding the information given above, the silicon dioxide content of raw potato is reported to be 10.1 mg/kg, and one litre of beer contains 131 mg.
* Refer to Document IIIA, section 2.10 for details of human risk assessment for silicon dioxide.

- In addition to the above, the potential for exposure to silicon dioxide when it is manufactured for use as an insecticide is minimal. Silicon dioxide is manufactured in a completely enclosed system, as is the manufacture of the insecticide product based on silicon dioxide. This means there is no exposure to workers, bystanders or the environment during manufacture. It is estimated that [redacted] of silicon dioxide will be manufactured each year for use as a biocide. This amount of silicon dioxide is tiny in comparison to the other non-biocidal uses of silicon dioxide. For example, amorphous silicon dioxide is the main component of glass and in 1995, 12.9 million tonnes of glass was discarded in the US alone.
  [redacted]

- Operator exposure work has been carried out in humans exposed to high concentrations of silicon dioxide. Such data has been used previously by a number of regulatory authorities to set national, international and supranational maximum exposure limits for safe working conditions, and all of these exposure limits are in general agreement. For example, the long term occupational exposure limit for silicon dioxide set in the UK is 2.4 mg/m^3 (respirable dust) (8h time weighted average). The US threshold limit value (TLV, set by the American Conference of Governmental Industrial Hygienists, ACGIH) for silicon dioxide is 2 mg/m^3 (respirable dust). In Australia, the long-term occupational exposure limit for silicon dioxide is also 2 mg/m^3 (respirable dust). The risk assessment for human exposure to silicon dioxide, when applying the representative product, RID Insect Powder shows that exposure to silicon dioxide does not exceed these agreed maximum exposure limits for safe working conditions*. As the objective of an animal test is to predict the toxicological effect in humans, then an established safe exposure level based on human data takes precedence over animal data generated for an approximation of a theoretical safe value.
  *The risk assessment for human exposure to silicon dioxide shows exposure to RID Insect Powder, under normal working conditions did not exceed the recommended UK maximum exposure limit to amorphous silicon dioxide (set at 2.4 mg/m^3 for respirable dust)**.
  ** Refer to Document IIIA, section 2.10 for details of human risk assessment for silicon dioxide.

- There is a substantial volume of information available for amorphous silicon dioxide. The data available are in general agreement, all showing that amorphous silicon dioxide *per se* is intrinsically biologically inert.

There is a substantial volume of information available for silicon dioxide, and while there are no studies available performed to specific guidelines, which consider chronic toxicity or carcinogenicity specifically, it does cover all the major biological considerations. Given the large volume of data available for silicon dioxide, only the typical findings have been summarised below with regards to the chronic toxicity and carcinogenic potential of silicon dioxide. A number of reviews have been conducted by different regulatory bodies including the EPA, and the FDA, who considered the health aspects of silicon dioxide as a food additive. EPA concluded that silicon dioxide's acute toxicity profile is characterised as moderate to low, and consequently silicon dioxide has been exempted from the requirement of a tolerance limit when applied to growing crops or agricultural commodities. FDA has classified silicon dioxide as Generally Recognised as Safe (GRAS) and has approved its use as a dietary food additive at levels of up to 2% by weight in food. The joint FAO/WHO Expert Committee evaluated a number of food additives. The anti-caking agent silicon dioxide was given an acceptable daily intake of "not limited". There are two FDA direct food ingredient regulations for silicon dioxide, plus a clearance by the US Department of Agriculture for its use in curing mixes and in animal feed premixes. In agreement with the review by the EPA, the FDA concluded that silicon dioxide appears to be biologically inert and there was no evidence available that suggests silicon dioxide is hazardous to humans.

Exposure to increasing concentrations of silicon dioxide: Effects and observations

Below is a summary of the long-term toxicity studies available for silicon dioxide. They are summarised in full under the relevant end points in Document IIIA.

Chronic, oral
Takizawa et al. orally administered 0, 0.125, 2.5 and 5% amorphous silica to $B_6C_3F_1$ mice and Fisher rats 93 weeks and 103 weeks respectively and found that repeated oral administration produced no significant treatment-related effects. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – Study summary 1 of 1).

Chronic, inhalation
Schepers exposed Wistar rats, guinea pigs and rabbits to 126 mg/m^3 amorphous silica by inhalation for a maximum of 24 months. No radiographic signs of lung disease in animals at the end of their maximal period of silicon dioxide inhalation were found. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – Study summary 1 of 2).

Choudat et al studied the health records and chest x-rays of 131 workers (male), 90 of which were the control group and 41 of which were the test group. The 41 men were exposed to 0 – 3.4 mg/m^3 respirable dust over a mean exposure period of 8 years. It was shown that the exposure to precipitated silica dust induces little respiratory impairment, which was increased by smoking. The test subject questionnaire, chest x-ray films and concentrations of arterial blood gas were used to distinguish the two groups of workers (exposed or not)

None of these methods were able to discriminate. Exposure to amorphous silica dust may induce a mild small airway disease, only in comparison to a control group. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.5 – study summary 2 of 2).

Repeated dose, inhalation
Reuzel et al. exposed Wistar rats to up to 30 mg/m^3 amorphous silica by inhalation for 90 days. It was found that amorphous silicas did not induce persistent granulomas and the adverse affects in the respiratory tract partly or completely regressed. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.4 – Study summary 1 of 2).

Johnston et al. exposed Fischer-344 rats to 50 mg/m^3 amorphous silica by inhalation for 90 days. It was found that amorphous silicon dioxide did not cause gene mutation, partly because of its low biopersistence and that the effects of exposure were reversible as demonstrated by the post-exposure results. (Referenced and summarised in Document IIIA, Annex point IIA, VI, 6.4 – Study summary 2 of 2).

Carcinogenicity
Takizawa et al. orally administered 0, 0.125, 2.5 and 5% amorphous silica to $B_6C_3F_1$ mice and Fisher rats 93 weeks and 103 weeks respectively and found that repeated oral administration produced no significant treatment-related effects. (Referenced and summarised in Document IIIA, Annex point IIA, 6.7 – Study summary 1 of 1).

Additionally, a review is available in the public domain of an unpublished study on the effects of amorphous silicon dioxide on multiple generations of rats. This study shows that there are no adverse effects to either generation when fed 100 mg/kg bw per day. Although this is an unpublished reference and therefore not useful for the risk assessment, it is considered suitable as supporting evidence for this data end point.

Conclusion
It has been demonstrated that the low level of exposure to silicon dioxide during its use as an insecticide (PT18) indicates that it is not scientifically necessary to conduct a multi-generations study on silicon dioxide as it will not add any useful information to the risk assessment. It has been shown in the human risk assessment that compared to exposures *via* the diet and the environment, exposure from silicon dioxide as an insecticide is insignificant. The risk assessment for human exposure to silicon dioxide, when applying the representative product RID Insect Powder shows that exposure to silicon dioxide does not exceed agreed, well established maximum exposure limits for safe working conditions with silicon dioxide and nuisance dust. The toxicological profile of silicon dioxide has been well established with a large body of data available in the public domain. The operator exposure limits that have been set for nuisance particles and dusts are also based on a large amount of available data. As shown above, data is available on the effects of exposure to amorphous silicon dioxide and this data shows that there are no lasting adverse effects. Although this data has its limitations and there are no studies available performed to specific guidelines which consider chronic toxicity or carcinogenicity, it is considered sufficient to address the toxicity of silicon dioxide particularly given the levels of exposure expected to silicon dioxide through other, non-biocidal uses of silicon dioxide including its use in food.

3.9 NEUROTOXICITY

Remark	Reference
It is not scientifically necessary to submit a neurotoxicity study for silicon dioxide, because the "Technical Guidance Document in Support of Directive 98/8/EC Concerning the Placing of Biocidal Products on the Market: Guidance on Data Requirements for Active Substances and Biocidal Products" states that this test is only required if there are any indications that the active substance may have neurotoxic properties. The safety profile of amorphous silicon dioxide is well established (see Document IIIA, Section 6.1.1 for further details) and there is a substantial volume of information available for silicon dioxide. The data available is in general agreement, all showing that amorphous silicon dioxide *per se* is intrinsically biologically inert. There is no data available which indicates that silicon dioxide may have neurotoxic properties. Generation of test data to determine neurotoxic effects of silicon dioxide is therefore not considered scientifically necessary.	Document IIIA, Section 6.9

3.10 HUMAN DATA

Effects of exposure to silicon dioxide in man are well reported in the product literature. This data has been summarised in Document IIIA, Sections 6.1.3, 6.4.3, 6.5 and 6.12. The key results for man include the following:

There is a substantial volume of data available on the toxicity of amorphous silicon dioxide, *via* both the oral and inhalation route. For man, acute oral LD_{50} has been estimated to be greater than 15000 mg/kg (see Document IIIA, Section 6.1.1. for further details).

As regards acute inhalation toxicity, even at the maximum attainable concentration in air (477 mgm^{-3}), no fatalities were caused amongst rats (see Document IIIA, Section 6.1.3 for further details).

There are no reported carcinogenic, species specific, reproduction, immunotoxic or hormone related effects for amorphous silicon dioxide. It is on this basis that it is not necessary to submit additional data regarding toxicity of amorphous silicon dioxide.

3.11 OTHER TOXICOLOGICAL EFFECTS

Remark	Reference
There are no reported toxicity effects of sufficient concern to justify further investigation by a mechanistic study. There are no reported carcinogenic, species specific, reproduction, immunotoxic or hormone related effects for amorphous silicon dioxide. It is on this basis that it is not necessary to submit additional data regarding any mechanism of silicon dioxide toxicity.	Document IIIA, Section 6.10
The major route of exposure to amorphous silicon dioxide from its use as an insecticide (PT18) is by inhalation. Therefore data on the parenteral administration of amorphous silicon dioxide has not been submitted.	Document IIIA, Section 6.11
It is not necessary to submit tests considering the toxicity of silicon dioxide in food and feeding stuffs because amorphous silicon dioxide as marketed by Rentokil Initial for use as an insecticide (PT18) is an approved food additive with Generally Regarded As Safe (GRAS) status. Therefore its exposure to food does not pose any risk.	Document IIIA, Section 6.14
Amorphous silicon dioxide is a stable compound (melting point >1500°C; solubility ≈100mg/L. Amorphous silicon dioxide has been shown not to produce any non mammalian-metabolite substances (see Document IIIA, Section A6.2 for further details). In addition, it has been shown that under test conditions, animals tested with amorphous silicon dioxide do not exhibit signs or symptoms suggesting interference with absorption of other dietary components as demonstrated by the lack of effect on food intake and weight gain (see Document IIIA, Section A6.1.1 and Section A6.5 for further details).	Document IIIA, Section 6.15
The main exposure to amorphous silicon dioxide is *via* the inhalation and the oral route. This will be equally true when amorphous silicon dioxide has been formulated for use as an insecticidal (PT18) powder. The main hazard will be from the inhalation of the dust from any such product. Effects of amorphous silicon dioxide dust inhalation have been widely reported along with the effects from exposure *via* the oral route. These have been examined fully in alternate sections of this dossier (Document IIIA, Section 6.1.3, Section 6.4.3, Section 6.1.1 etc.). Therefore it is not deemed necessary to perform any other tests related to the exposure of humans as this will not add anything useful to the risk assessment.	Document IIIA, Section 6.16 Document IIIA, Section 6.17
Therefore it is not deemed necessary to generate further data as it will not provide any useful information for the risk assessment.	
It is not necessary to provide data on the toxic effect of metabolites from treated plants because silicon dioxide is not intended for use directly on plants or plant products.	

4 ENVIRONMENTAL EFFECTS ASSESSMENT

4.1 FATE AND DISTRIBUTION IN THE ENVIRONMENT

4.1.1 Degradation

4.1.1.1 Biodegradation (1 of 2)

Guideline / Test method	Test type	Test parameter	Inoculum Type	Inoculum Conc	Inoculum Adaptation	Additional substrate	Test substance conc.	Degradation Incubation period	Degradation Degree [%]	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Ready Biodegradability: Silicon dioxide is an inorganic chemical but the approved EC method C4 (a –f) applies only to organic compounds and the "TNsG" state that the ready biodegradation test is required only of organic compounds. Therefore a ready biodegradation test for silicon dioxide has not been submitted.	Document IIIA, Section 7.1.1.2.1
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Inherent Biodegradability: Silicon dioxide is an inorganic chemical but the approved EC methods C9 and C12 apply only to water-soluble, non-volatile organic substances. Therefore an inherent biodegradation test for silicon dioxide has not been submitted.	Document IIIA, Section 7.1.1.2.2
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Biodegradation in sea water: Silicon dioxide is an inorganic chemical but the process applies only to organic compounds. Silicon dioxide is not intended to be either used or released into marine environments. Therefore a biodegradation test for silicon dioxide in seawater has not been submitted.	Document IIIA, Section 7.1.1.2.3
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Biological sewage treatment – aerobic biodegradation: Silicon dioxide is an inorganic chemical but the process applies only to organic compounds. Therefore a test to determine the aerobic biodegradation of silicon dioxide in sewage has not been submitted.	Document IIIA, Section 7. 1.2.1.1
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Biological sewage treatment – anaerobic biodegradation: Silicon dioxide is an inorganic chemical but the process applies only to organic compounds. Silicon dioxide is not intended to be exposed to anaerobic conditions, such as manure storage facilities in animal housing. Therefore a test to determine the anaerobic biodegradation of silicon dioxide in sewage has not been submitted.	Document IIIA, Section 7.1.2.1.2

4.1.1.1 Biodegradation (2 of 2)

Guideline / Test method	Test type	Test parameter	Inoculum Type	Inoculum Conc	Inoculum Adaptation	Additional substrate	Test substance conc.	Degradation Incubation period	Degradation Degree [%]	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Biodegradation in freshwater – aerobic aquatic degradation study: Silicon dioxide is an inorganic chemical but the approved EC methods for ready biodegradability (EC method C4 a-f) apply only to organic compounds and the "TNsG" state that the ready biodegradation test is required of organic compounds. Also the approved EC test methods C9 and C12 are designed to work with water-soluble, non-volatile organic substances. Therefore an aerobic aquatic biodegradation study for silicon dioxide has not been submitted.	Document IIIA, Section 7.1.2.2.1
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Biodegradation in freshwater – water/sediment degradation study Under normal conditions of use silicon dioxide will not be applied directly or indirectly to the sediment in aquatic systems. In addition: silicon dioxide is an inorganic chemical and the approved EC methods for ready biodegradability (EC method C4 a-f) apply only to organic compounds. Also the "TNsG" state that the ready biodegradation test is required of organic compounds. Inherent biodegradability (A7.1.1.2.2) is technically not feasible as the approved EC test methods C9 and C12 are designed to work with water-soluble, non-volatile organic substances. Therefore a study to determine the biodegradation of silicon dioxide in freshwater/sediment has not been submitted.	Document IIIA, Section 7.1.2.2.2

Footnotes

1. It is not considered necessary to determine rate and route of degradation in aquatic systems including the identification of metabolites (Document IIIA, 7.1.2) for the following reasons:

a) Testing for the ready biodegradability (Document IIIA, Section A7.1.1.2.1) of silicon dioxide is scientifically unjustified. Silicon dioxide is an inorganic chemical, with the molecular formula O=Si=O. It is scientifically not necessary to determine the biodegradability of inorganic chemicals, because the approved EC method for ready biodegradability (EC method C4 a-f) applies only to organic compounds. In addition, the "Technical Guidance Document in Support of Directive 98/8/EC Concerning the Placing of Biocidal Products on the Market: Guidance on Data Requirements for Active Substances and Biocidal Products" states that the ready biodegradation test is required of organic compounds.

b) Inherent biodegradability (Document IIIA, Section A7.1.1.2.2) is technically not feasible to perform on silicon dioxide as the approved EC test methods C9 and C12 are designed to work with water-soluble, non-volatile organic substances. While silicon dioxide is slightly soluble and non-volatile, it is an inorganic compound.

Notwithstanding the above, the preliminary risk assessment for exposure to water does not indicate the need to conduct additional studies on the fate and behaviour of silicon dioxide in the aquatic compartment.

It is for the reasons given above that additional test data about the degradation of silicon dioxide in aquatic systems has not been submitted.

4.1.1.2 Abiotic Degradation

Hydrolysis

Guideline /Test Method	pH	Temperature [°C]	Initial TS concentration C_0 [mol/l]	Reaction rate Constant, K_h [1/s x 10^5]	Half-life, DT_{50} [h]	Coefficient of correlation, r_2	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	OECD Method 111: Hydrolysis as a function of pH states that the method is applicable only to substances for which the analytical method has sufficient accuracy [to detect >10% hydrolysis]. For silicon dioxide to be analysed in this test, it would involve colorimetry and would require the use of pH buffered solutions. Immediately the colorimetric solutions are prepared, the pH is altered, all silicon species that are present will be changed back to silicon dioxide at that pH. Therefore, the analysis of any change in silicon dioxide content of the test solutions is impossible. Considering the above arguments, it is not deemed possible to perform this test.	Document IIIA, Section 7.1.1.1.1

Photolysis in water

Guideline/ Test Method	Initial Molar TS concentration	Total Recovery of Test Substance [% of appl. a.s.]	Photolysis rate constant (k^c_p)	Direct photolysis sunlight rate constant (K_{pE})	Reaction quantum yield (0^c_E)	Half-life ($t_{1/2E}$)	Remarks	Reference
OECD Guidelines for the Testing of Chemicals. Proposal for a New Guideline, Phototransformation of Chemicals in Water – Direct and Indirect Photolysis. Draft document August 2000	N/A	N/A	N/A	$K_{d(max)}$: The average maximum rate constants for the two replicates were 16 and 3 day^{-1} for summer and winter conditions at 50°N respectively.	N/A	N/A	The first tier test performed in this study is considered to have met all validity criteria. Given the estimated half-lives given above, calculations suggest that the test substance photolyses rapidly in both summer and winter conditions at 50°N. According to the OECD guideline this substance would be expected to proceed to further testing. However, it is felt that the calculations do not give a realistic estimate of photolysis for this substance. Firstly the absorbance and molar extinction coefficients above 295 nm are very low, such that the test substance would not be expected to photolyse. Secondly the calculations assume that the test substance absorbs every photon of light, ie the quantum yield is equal to 1. In reality the quantum yield is generally much less than 1 (usually <0.1 and sometimes <0.01). The maximum rate constant, as determined by further testing would therefore be considered to be slower. A further consideration is that in order to perform the full study the concentration of the test substance must be measured. In the absence of a method able determine silicon dioxide (to determine measured concentrations in other studies on this substance silicon levels were measured), it would be impossible to determine losses of the parent. It was therefore considered inappropriate to perform further testing as the study is technically not possible to perform under guidance from the Biocidal Products Directive.	Document IIIA, Section 7.1.1.1.2

Phototransformation in air

Guideline / Test Method	Initial Molar TS concentration	Total Recovery of Test Substance [% of appl. a.s.]	Photolysis rate constant (k^c_p)	Direct photolysis sunlight rate constant (K_{pE})	Reaction quantum yield (0^c_E)	Half-life ($t_{1/2E}$)	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	Phototransformation in air (estimation method), including identification of breakdown products: Silicon dioxide is not volatile, and therefore exposure via the atmospheric compartment is not considered relevant. Notwithstanding the above, the structure of silicon dioxide is O=Si=O. This structure means that OH-radicals are unlikely to be generated during degradation in air. When pseudo-first order rate constant for degradation in air was estimated using the QSAR method, the rate constant was zero. This result supports the above statement that OH radicals are unlikely to be generated during degradation of silicon dioxide in air. Silicon dioxide will not have an impact on global warming because it does not exist in the gaseous state at ambient temperature and pressure. The presence of absorption bands in the IR spectrum region 800-1200nm is therefore not applicable. It is also highly unlikely that silicon dioxide will have any impact either on ozone depletion in the stratosphere or ozone formation in the troposphere. This is because silicon dioxide does not contain chlorine substituents, and OH radicals are unlikely to be generated during degradation of silicon dioxide in air. The final atmospheric risk indicator is acidification. As silicon dioxide does not contain Cl, F, N or S substituents, acidification is not considered to be a risk to receiving soil or surface water.	Document IIIA, Section 7.3.1

Footnote

Further studies to determine fate and behaviour of silicon dioxide in the air (Document IIIA, Section 7.3.2) are not considered necessary because The "TNsG" states that further studies are required to determine fate and behaviour in air if: The active substance is to be used in fumigant preparations and the active substance causes risk to the atmospheric compartment. Silicon dioxide is not intended for use as a fumigant. As shown in Document IIIA, Section 7.3.1 the preliminary risk assessment for exposure to the atmosphere does not indicate the requirement for additional studies. Therefore further studies have not been submitted.

4.1.1.3 Distribution

Absorption onto/desorption from soils (1 of 4)

Guideline /test method	Absorbed a.s. [%]	K_a [1]	K_{aOC} [2]	K_d [3]	K_{dOC} [4]	K_a / K_d [5]	Degradation products		Remarks	Reference
							Name	[%] of a.s.		
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Amorphous silicon dioxide is not expected to reach the soil compartment (see Document IIIA, Section 2.10 for exposure assessment) and there are no indications that it will bioaccumulate (see Document IIIA, Section 7.4.2 and Section 2.10 for further details). Also a value for log K_{oc} can be calculated. In the *Technical Guidance Document on Risk Assessment in support of Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market*, (TGD) Chapter 3, it states that K_{oc} can be estimated using K_{ow} for non-ionic substances using QSARS. Log K_{ow} has been calculated for silicon dioxide to be 0.53 (see Document IIIA, Section 3.9 for calculation). Using LOGKOW as the most appropriate QSAR from Table 4, Page 26, Chapter 4 of the TGD gives the equation for the estimation of log K_{oc} for a non-hydrophobic substance as: log K_{oc} = 0.52 log K_{ow} + 1.02 Therefore for silicon dioxide: log K_{oc} = (0.52 x 0.53) + 1.02 = 1.30 and a standard error of 0.56 giving: log K_{oc} = 1.30 ± 0.56. As this calculation is expected to reflect a result determined by experimentation, it is not deemed scientifically necessary to perform any further studies.	Document IIIA Section 7.1.3 Document IIIA Section 7.1.4 Document IIIA Section 7.1.4.1

Footnotes

1. It is not scientifically necessary to conduct further studies on the adsorption and desorption of amorphous silicon dioxide in water sediment systems (Document IIIA, Section 7.1.4) because the preliminary risk assessment indicates it is scientifically unjustified, and not necessary due to prerequisites fulfilled on limited exposure and toxicity profile.

Amorphous silicon dioxide does not biodegrade (refer to data end points Document IIIA, Section 7.1.1.2.1 and Document IIIA, Section 7.1.1.2.2). Notwithstanding this, it is not scientifically necessary to determine the aerobic biodegradation of amorphous silicon dioxide in soil due to prerequisites fulfilled on limited exposure and toxicity profile. This is because: a. Amorphous silicon dioxide as used as an insecticide (PT18) is intended for indoor use only. b. Amorphous silicon dioxide as used as an insecticide (PT18) is not intended for direct application to the environment c. Notwithstanding the above, there is potential for exposure to the environment as a result of disposal of waste material. The risk to the environment from the act of disposal is considered to be insignificant. This is because the quantity of amorphous silicon dioxide being disposed of compared to the volume of total waste is minute. The total estimated disposal of amorphous silicon dioxide across the whole of the EU is < 0.00000073% of the total waste generated and sent to landfill in the UK alone (see Document IIA, Section 2.10 for further details). This means that any amorphous silicon dioxide that is sent for landfill is massively diluted by the large volume of municipal waste continually entering landfill sites in the UK. The data available on the environmental toxicity of amorphous silicon dioxide shows that this volume is extremely unlikely to cause any adverse effect to the environment, and as such requires no further investigation.

2. A field study on accumulation in sediment (Document IIIA, Section 7.1.4.1) has not been submitted for the following reasons: The "TNsG" states that a field trial on accumulation in the sediment is needed only if non extractable residues are formed in the initial water/sediment study submitted in Document IIIA, section 7.1.2.2.2, and these residues exceed 70% of the initial dose, or if the mineralisation rate in this study is less than 5% in 100 days. An initial water/sediment study (Document IIIA, Section 7.1.2.2.2) has not been submitted in, for the following reasons: a. The testing of the biodegradation of silicon dioxide in freshwater/sediment is scientifically unjustified because silicon dioxide, under normal conditions of use in Rentokil Initial's insecticide (PT18) products will not be applied directly or indirectly to the sediment in aquatic systems. b. Testing for the ready biodegradability (Document IIIA, Section A7.1.1.2.1) of silicon dioxide is scientifically unjustified. Silicon dioxide is an inorganic chemical, with the molecular formula O=Si=O. It is scientifically not necessary to determine the biodegradability of inorganic chemicals, because the approved EC method for ready biodegradability (EC method C4 a-f) applies only to organic compounds. In addition, the "TNsG" states that the ready biodegradation test is required of organic compounds. c. Inherent biodegradability (Document IIIA, Section A7.1.1.2.2) is technically not feasible to perform on silicon dioxide as the approved EC test methods C9 and C12 are designed to work with water-soluble, non-volatile organic substances. While silicon dioxide is slightly soluble and non-volatile, it is an inorganic compound. It is for the same reasons that it is not necessary to submit a field trial on accumulation of silicon dioxide in the sediment. Notwithstanding this, the preliminary risk assessment for exposure to water does not indicate the need to conduct additional studies on the fate and behaviour of silicon dioxide in the aquatic compartment.

Absorption onto/desorption from soils (2 of 4)

Guideline/ test method	Absorbed a.s. [%]	K_a [1]	K_{aOC} [2]	K_d [3]	K_{dOC} [4]	K_a / K_d [5]	Degradation products Name	[%] of a.s.	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Aerobic degradation in soil, initial study: Silicon dioxide is an inorganic chemical, with the molecular formula O=Si=O. The approved test guideline OECD 304A applies only to ^{14}C-labelled material. Therefore a test to determine the aerobic biodegradation of silicon dioxide in soil has not been submitted.	Document IIIA Section 7.2.1
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Aerobic degradation in soil, further studies: Silicon dioxide is an inorganic chemical, with the molecular formula O=Si=O and biodegradability is relevant only to organic compounds. Therefore tests to determine the aerobic degradation of silicon dioxide in soil have not been submitted. Notwithstanding this, the preliminary risk assessment for exposure to soil does not indicate the need to conduct additional studies on the fate and behaviour of silicon dioxide in the soil compartment.	Document IIIA Section 7.2.2
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Rate and route of degradation: The "TNsG" states that the rate and route of degradation including the identification of any metabolites and degradation products in at least three soil types under appropriate conditions is required only if: a. The DT_{50lab} determined in the initial aerobic degradation study in soil (Document IIIA, section 7.2.1) is more than 21 days and the PEC/PNEC >1 for soil; b. there is danger for groundwater; c. other refinement of the preliminary risk assessment for soil is necessary. Silicon dioxide is an inorganic chemical, with the molecular formula O=Si=O and the approved test guideline OECD 304A applies only to ^{14}C-labelled material. Notwithstanding the above, the preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment. Therefore an initial aerobic degradation study in soil has not been submitted.	Document IIIA Section 7.2.2.1

Absorption onto/desorption from soils (3 of 4)

Guideline/ test method	Absorbed a.s. [%]	K_a [1]	K_{aOC} [2]	K_d [3]	K_{dOC} [4]	K_a / K_d [5]	Degradation products		Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Field soil dissipation and accumulation: The "TNsG" states that field soil dissipation and accumulation are required in two soil types if a. The $DT_{90field}$ is over one year and; b. The $DT_{50field}$ is greater than 3 months or; c. If during laboratory tests non-extractable residues are formed in amounts exceeding 70% of the initial dose after 100 days with a mineralization rate of less than 5% in 100 days. Silicon dioxide has the molecular formula O=Si=O and the approved test guideline OECD 304A applies only to ^{14}C-labelled material. Notwithstanding the above, the preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment, and therefore it is not considered necessary to submit additional data on field soil dissipation and accumulation. Therefore an initial aerobic degradation study in soil has not been submitted.	Document IIIA Section 7.2.2.2
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Extent and nature of bound residues: The "TNsG" states that data on the extent and nature of bound residues on soil are required if data submitted in Document IIIA, Section A7.2.1 and A7.2.2.1 indicate that bound residues may be formed which account for more than 10% of the active substance added. This end point is not relevant for silicon dioxide, on the basis on data submitted in Document IIIA, Section A7.2.1 and A7.2.2.1.	Document IIIA Section 7.2.2.3

Absorption onto/desorption from soils (4 of 4)

Guideline/ test method	Absorbed a.s. [%]	K_a [1]	K_{aOC} [2]	K_d [3]	K_{dOC} [4]	K_a / K_d [5]	Degradation products		Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Other soil degradation studies: The preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment, and therefore it is not considered necessary to submit additional data on release to soil under different release conditions.	Document IIIA Section 7.2.2.4
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Adsorption and mobility in soil further studies: The preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment, and therefore it is not considered necessary to submit additional data on adsorption and mobility studies in soil.	Document IIIA Section 7.2.3
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Adsorption and desorption in accordance with the new test guideline EC C18 or the corresponding OECD 106 and, where relevant, adsorption and desorption metabolites and degradation products: Silicon dioxide, under normal conditions of use in Rentokil Initial's insecticide (PT18) products, will not be applied directly on soil or released to soil in relevant concentrations. The preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment, and therefore it is not considered necessary to submit additional data on adsorption and mobility studies in soil.	Document IIIA Section 7.2.3.1
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Mobility in at least three soil types and where relevant mobility of metabolites and degradation products: The preliminary risk assessment for exposure to soil does not indicate the need to conduct studies on the fate and behaviour of silicon dioxide in the soil compartment, and therefore it is not considered necessary to submit additional data on mobility of silicon dioxide in soil.	Document IIIA Section 7.2.3.2

Key

1. K_a = Adsorption coefficient.
2. K_{aOC} = Adsorption coefficient based on organic carbon content.
3. K_d = Desorption coefficient.
4. K_{dOC} = Desorption coefficient based on organic carbon content.
5. K_a/K_d = Adsorption/desorption distribution coefficient.

4.1.2 Accumulation

Measurements of aquatic bioconcentration

Guideline /Test method	Exposure	Log Pow of a.s.	Initial concentration of a.s.	Steady-state BCF	Uptake rate constant	Depuration rate constant	Depuration time (DT_{50})	Metabolites	Remarks	Reference
N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	"Bioconcentration" is the process leading to a higher concentration of, for example, a pesticide in an organism than in environmental media to which it is exposed. The "Technical Guidance Document in support of Commission Directive 93.67/EEC on risk assessment for new notified substances and Commission Regulation EC No 1488/94 on risk assessment for existing substances. Part II Environmental risk assessment" states that the following are indicators of bioaccumulation potential a. if the substance has a partition coefficient log Kow ≥ 3 or; b. the substance is highly adsorptive or; c. the substance belongs to a class of substances known to have a potential to accumulate in living organisms or; d. there are indications from structural features. From the data available, silicon dioxide is not expected to have an intrinsic potential for bioconcentration in aquatic organisms, on the basis that it has an estimated partition coefficient of 0.53 (refer to Document IIIA, Section 3.9 for detail).	Document IIIA Section 7.4.2

Estimations on aquatic bioconcentration

Basis for estimation	Log Pow (measured)	Estimated BCF for fish (freshwater)	Estimated BCF for fish eating bird/predator	Remarks	Reference
N/A	N/A	N/A	N/A	"Bioconcentration" is the process leading to a higher concentration of, for example, a pesticide in an organism than in environmental media to which it is exposed. The "Technical Guidance Document in support of Commission Directive 93.67/EEC on risk assessment for new notified substances and Commission Regulation EC No 1488/94 on risk assessment for existing substances. Part II Environmental risk assessment" states that the following are indicators of bioaccumulation potential a. if the substance has a partition coefficient log Kow ≥ 3 or; b. the substance is highly adsorptive or; c. the substance belongs to a class of substances known to have a potential to accumulate in living organisms or; d. there are indications from structural features. From the data available, silicon dioxide is not expected to have an intrinsic potential for bioconcentration in aquatic organisms, on the basis that it has an estimated partition coefficient of 0.53 (refer to Document IIIA, Section 3.9 for detail).	Document IIIA Section 7.4.2

Estimation on terrestrial bioconcentration

Basis for estimation	Log Pow (measured)	Estimated BCF for				Remarks	Reference
		Terrestrial food chain I		Terrestrial food chain II			
		Soil dwelling species	Predatory bird/ vertebrate	Terrestrial plant	Grazing non-target organism		
N/A	N/A	N/A	N/A	N/A	N/A	"Bioconcentration" is the process leading to a higher concentration of, for example, a pesticide in an organism than in environmental media to which it is exposed. The "Technical Guidance Document in support of Commission Directive 93.67/EEC on risk assessment for new notified substances and Commission Regulation EC No 1488/94 on risk assessment for existing substances. Part II Environmental risk assessment" states that the following are indicators of bioaccumulation potential a. if the substance has a partition coefficient log Kow ≥ 3 or; b. the substance is highly adsorptive or; c. the substance belongs to a class of substances known to have a potential to accumulate in living organisms or; d. there are indications from structural features. From the data available, silicon dioxide is not expected to have an intrinsic potential for bioconcentration in aquatic organisms, on the basis that it has an estimated partition coefficient of 0.53 (refer to Document IIIA, Section 3.9 for detail).	Document IIIA Section 7.5.5

Footnote
The "Technical Guidance Document in Support of Directive 98/8/EC Concerning the Placing of Biocidal Products on the Market: Guidance on Data Requirements for Active Substances and Biocidal Products" states that a test on bioconcentration in earthworms is required if the risk assessment for secondary poisoning would suggest a concern for predators (Document IIA, Section 7.5.5.1). The environmental risk assessment for silicon dioxide shows there is no risk of secondary poisoning under normal conditions of use in Rentokil Initial's insecticide (PT 18) products. As there is no concern for predators, the test to determine bioconcentration of silicon dioxide in earthworms is not considered necessary.

4.2 EFFECT ON ENVIRONMENTAL ORGANISMS

4.2.1 Aquatic compartment

Acute toxicity to fish

Guideline/ Test method	Species	Endpoint/ Type of test	Exposure Design	Duration	Results LC_0 LC_{50} LC_{100}	Remarks	Reference
OECD Guidelines for Testing of Chemicals. Method 203. Fish, Acute Toxicity Test. Adopted 17 July 1992.	*Oncorhynchus mykiss*	Acute toxicity	The test procedure employed was a static system. Single borosilicate glass vessels (external dimensions; 460 mm × 305 mm × 310 mm; length × width × height) were used for the dilution water control and the exposure solution. The vessels had a working volume of 25 L. The test was undertaken in a temperature controlled room which was set at the nominal test temperature of 15 ± 1°C. The test solutions were gently aerated. The photoperiod in this study was 16 hours fluorescent light and 8 hours dark with 20 minute dawn and dusk transition periods commencing at 06:00 and 21:40 hours. At the start of the test ten fish were randomly allocated to the single test concentration and the dilution water control. The fish were not fed during the course of the test.	96 h	NOEC = 110 mg/L	There were no mortalities at the limit of solubility for amorphous silicon dioxide.	Document IIIA, Section 7.4.1.1

Footnotes

1. Due to the results available on the acute toxicity of silicon dioxide to fish, coupled with the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide to aquatic organisms (the data requirements detailed in Document IIIA, Section 7.4.3). It is also not necessary to submit data on prolonged toxicity of silicon dioxide to fish (Document IIIA, Section 7.4.3.1).

2. Due to the results available in the core base set of environmental toxicity data for silicon dioxide, particularly the lack of acute toxicity to fish and the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide on the reproduction and growth rate of fish (the data requirements detailed in Document IIIA, Section 7.4.3.2).

3. Due to the fact that there is no exposure to the aquatic environment, coupled with the fact that there is no data available which suggests that silicon dioxide will bioaccumulate in the environment, nor is there a risk of secondary poisoning through the use of silicon dioxide, it is not necessary to submit data on bioaccumulation in fish (the data requirements detailed in Document IIIA, Section 7.4.3.3.1).

Acute toxicity to invertebrates

Guideline/ Test method	Endpoint / Type of test	Exposure Design	Duration	Results LC_0 LC_{50} LC_{100}	Remarks	Reference
OECD Guidelines for the Testing of Chemicals. Test Guideline 202 Part I, Daphnia sp., Acute Immobilisation Test. Adopted 4 April 1984.	Acute toxicity	Borosilicate glass beakers of 250 ml nominal capacity were used as test vessels, with four replicates for the dilution water control and exposure solution. Each vessel contained 200 ml of test solution providing a depth of approximately 60 mm. The beakers were covered with loose fitting glass lids. The positions of the treatments were randomly allocated within the test area. The test was initiated by the addition of five randomly selected *Daphnia*, in <2.0 ml of dilution water, to each test vessel. The dilution water control and exposure solution contained a total of 20 *Daphnia*. The loading of the *Daphnia* in each test vessel was 25 *Daphnia*/L. The nominal test solution temperature was 20 ± 1°C, maintained by control of the room temperature. A photoperiod of 16 hours light:8 hours dark, with 20 minute dusk and dawn transition periods, was provided. The test solutions were not aerated and the *Daphnia* were not fed during the course of the study.	48 h	NOEC = 86 mg/L	No symptoms of toxicity were observed in this study.	Document IIIA, Section 7.4.1.2

Footnotes

1. Due to the results available on the acute toxicity of silicon dioxide to *Daphnia magna*, coupled with the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide to aquatic organisms (the data requirements detailed in Document IIIA, Section 7.4.3).
2. Due to the fact that there is no exposure to the aquatic environment, coupled with the fact that there is no data available which suggests that silicon dioxide will bioaccumulate in the environment, nor is there a risk of secondary poisoning through the use of silicon dioxide, it is not necessary to submit data on bioaccumulation in invertebrate species (the data requirements detailed in Document IIIA, Section 7.4.3.3.2).
3. Due to the results available in the core base set of environmental toxicity data for silicon dioxide, particularly that available on the acute toxicity to *Daphnia magna* and the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide on the reproduction and growth rate of invertebrates (the data requirements detailed in Document IIIA, Section 7.4.3.4).

Growth inhibition on algae

Guideline / Test method	Species	Endpoint / Type of test	Exposure Design	Duration	Results NOE_rC	Results E_bC_{50} [1]	Results E_rC_{50} [2]	Remarks	Reference
OECD Guidelines for Testing of Chemicals. Test Guideline 201. Alga, Growth Inhibition Test. Adopted 7 June 1984.	*Selenatrum capricornutum*	Growth inhibition test	The test vessels were borosilicate glass conical flasks of 250 ml nominal capacity closed with polyurethane foam bungs. Each flask contained 100 ml of test solution. The cultures were incubated at 24 ± 2°C (the nominal test temperature), under continuous "cool-white" illumination, with orbital shaking at 160 rpm, in a Gallenkamp type INR-401 orbital incubator. Six replicate cultures of the culture medium control and single concentration of test substance were employed. The positions of the test vessels in the incubator were randomised by rows, and re-randomised daily. One blank vessel (without algal inoculum) for the culture medium control and each test concentration was incubated concurrently. The algal cell densities of the inoculum and test cultures were determined by electronic particle counting, using a Coulter counter model Z1, counting at a lower threshold equivalent spherical diameter of approximately 2.3 µm. Each replicate test vessel was inoculated with 0.79 ml of the inoculum culture to give a nominal cell density of 1.00×10^4 cells/mL. Three 100 ml volumes of Coulter electrolyte, inoculated in the same manner, had a mean measured cell density of 1.01×10^4 cells/mL. The latter value was used for growth calculations. After 24, 48 and 72 hours, (1, 2 and 3 days) samples were removed from each test and blank vessel. The appropriate blank particle count was subtracted from that of the test culture to obtain the cell density.	72 h	54 mg/L	> 54 mg/L	> 54 mg/L	No inhibition was observed.	Document IIIA, Section 7.4.1.3

Key

1. Calculated from the area under the growth curve
2. Calculated from growth rate

Footnotes

1. Due to the results available on the toxicity of silicon dioxide to algae, coupled with the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide to aquatic organisms (the data requirements detailed in Document IIIA, Section 7.4.3).

Inhibition of microbial activity (aquatic)

Guideline / Test method	Species / Inoculum	Endpoint/ Type of test	Exposure		Results			Remarks	Reference
			Design	Duration	EC_{20}	EC_{50}	EC_{80}		
OECD Test Guideline 209, Activated Sludge, Respiration Inhibition Test. Adopted 4 April 1984.	Activated sludge from [redacted] treating sewage of predominantly domestic origin.	Respiration inhibition	This test measures the respiration rate of an activated sludge 3 hours after feeding an excess, but standard amount, of a synthetic sewage and compares this with the respiration rate of the same activated sludge in the presence of the test chemical. 3,5-dichlorophenol is used as a reference substance as it has known inhibitory effects on respiration and ensures that the batch of sludge used in the test shows a normal level of sensitivity. A single nominal 1000 mg/L concentration of test substance was prepared in duplicate together with three control culture flasks. Four flasks containing the reference substance, 3,5-dichlorophenol, at nominal concentrations of 3.2, 10, 32 and 100 mg/L were also prepared. In addition a single abiotic flask containing 100 mg/L 3,5-dichlorophenol but no activated sludge was prepared. Each flask contained an excess of the synthetic sewage, sufficient activated sludge to give final solids concentrations of 1600 mg/L, an appropriate quantity of either test substance or 3,5-dichlorophenol stock solution and aerated water to give a final flask contents volume of 500 ml. The exact quantities of each of these constituents are given in Table A7_4_1_4-5. The pH of each flask was measured before the start of the test. Flasks were set up in batches of six and aerated at 20 ± 2°C for 3 hours. Each batch included a control flask and five test or reference substance flasks. The temperatures of the flask contents were measured at the end of the 3 hours aeration using a mercury-in-glass thermometer.	3 h	>1000 mg/L	>1000 mg/L	>1000 mg/L	No inhibition occurred due to application of the test material. NOEC = 1000 mg/L	Document IIIA, Section 7.4.1.4

Footnotes

1. Due to the results available on the toxicity of silicon dioxide to microbes, coupled with the fact that there is no exposure to the aquatic environment, it is not necessary to submit further studies on the effects of silicon dioxide to aquatic organisms (the data requirements detailed in Document IIIA, Section 7.4.3).

Effects on sediment dwelling organisms

Remarks	Reference
The "Technical Guidance Document in Support of Directive 98/8/EC Concerning the Placing of Biocidal Products on the Market: Guidance on Data Requirements for Active Substances and Biocidal Products" states that this information is only required if the active substance partitions to, and persists in, aquatic sediments such that sediment dwelling organisms are likely to be exposed to the active substance. The core base data set for silicon dioxide does not indicate that silicon dioxide poses a danger to sediment dwelling organisms. In addition, the environmental risk assessment for silicon dioxide shows that no exposure of sediment dwelling organisms is expected under normal conditions of use in Rentokil Initial's insecticide (PT18) products. It is for these reasons that a study to determine the effects of silicon dioxide on sediment dwelling organisms has not been submitted.	Document IIIA, Section 7.4.3.5.1

Aquatic Plant Toxicity

Remarks	Reference
The "Technical Guidance Document in Support of Directive 98/8/EC Concerning the Placing of Biocidal Products on the Market: Guidance on Data Requirements for Active Substances and Biocidal Products" states that further studies on the effects on aquatic organisms, such as aquatic plants, are required only if the results of data submitted for the end points in Document IIIA, Sections 7.4.1.1, 7.4.1.2, 7.4.1.3 and 7.4.1.4 indicate a danger to the environment. As the results of the tests submitted for the end points in Document IIIA, Sections 7.4.1.1, 7.4.1.2, 7.4.1.3 and 7.4.1.4 do not indicate that silicon dioxide poses a danger to the environment, it is not considered necessary to submit data that considers toxicity of silicon dioxide to aquatic plants.	Document IIIA, Section 7.4.3.5.2

4.2.2 Atmosphere

Silicon dioxide is not volatile, and therefore exposure via the atmospheric compartment is not considered relevant.
Notwithstanding the above, the structure of silicon dioxide is O=Si=O. This structure means that OH-radicals are unlikely to be generated during degradation in air. When pseudo-first order rate constant for degradation in air was estimated using the QSAR method, the rate constant was zero. This result supports the above statement that OH radicals are unlikely to be generated during degradation of silicon dioxide in air.
Silicon dioxide will not have an impact on global warming because it does not exist in the gaseous state at ambient temperature and pressure. The presence of absorption bands in the IR spectrum region 800-1200nm is therefore not applicable. It is also highly unlikely that silicon dioxide will have any impact either on ozone depletion in the stratosphere or ozone formation in the troposphere. This is because silicon dioxide does not contain chlorine substituents, and OH radicals are unlikely to be generated during degradation of silicon dioxide in air. The final atmospheric risk indicator is acidification. As silicon dioxide does not contain Cl, F, N or S substituents, acidification is not considered to be a risk to receiving soil or surface water.

4.2.3 Terrestrial compartment

Toxicity to terrestrial organisms, initial tests (1 of 2)

Guideline /Test method	Species	Endpoint / Type of test	Exposure		Results			Remarks	Reference
			Design	Duration	NOEC	LOEC	EC/ LC_{50}		
N/A	Microbes, terrestrial	Inhibition	N/A	N/A	N/A	N/A	N/A	The environmental risk assessment for silicon dioxide does not indicate that it poses a risk to the terrestrial compartment. Therefore it is not considered necessary to submit data on the effect of silicon dioxide on the inhibition of microbial activity in the terrestrial compartment.	Document IIIA, Section 7.5.1.1
N/A	Earthworm	Acute toxicity	N/A	N/A	N/A	N/A	N/A	The information on the environmental exposure scenario for silicon dioxide (as given in Document IIIB, Section 7.1) does not indicate that it poses a risk to the terrestrial compartment. Therefore it is not considered necessary to submit data on the acute toxicity of silicon dioxide to earthworms.	Document IIIA, Section 7.5.1.2
N/A	Plants	Acute toxicity	N/A	N/A	N/A	N/A	N/A	The environmental risk assessment for silicon dioxide does not indicate that it poses a risk to the terrestrial compartment. It is on this basis that it is not considered necessary to submit data on the acute toxicity of silicon dioxide to plants.	Document IIIA, Section 7.5.1.3
N/A	Birds	Acute oral Short term Reproduction	N/A	N/A	N/A	N/A	N/A	Silicon dioxide, under normal conditions of use in Rentokil Initial's insecticide (PT 18) products will be applied indoors only. Data submitted in Document IIIA, section 7.4.2 shows that silicon dioxide is not expected to have an intrinsic potential for bioaccumulation. The environmental risk assessment for silicon dioxide shows there is no risk of secondary poisoning under normal conditions of use in Rentokil Initial's insecticide (PT 18) products. There is no data available to suggest that silicon dioxide is hazardous to birds. Therefore studies determining the acute oral toxicity, short term toxicity and effects on reproduction of silicon dioxide to birds have not been submitted.	Document IIIA, Sections 7.5.3.1.1 7.5.3.1.2 7.5.3.1.3
N/A	Honeybee and other beneficial arthropods	Acute toxicity	N/A	N/A	N/A	N/A	N/A	As silicon dioxide, under normal conditions of use in Rentokil Initial's insecticide (PT 18) products will be applied indoors only, it is not considered necessary to conduct this test.	Document IIIA, Section 7.5.4.1

Toxicity to terrestrial organisms, initial tests (2 of 2)

Guideline /Test method	Species	Endpoint / Type of test	Exposure Design	Exposure Duration	Results NOEC	Results LOEC	Results EC/ LC_{50}	Remarks	Reference
N/A	Other terrestrial non-target organism	N/A	N/A	N/A	N/A	N/A	N/A	The environmental risk assessment for silicon dioxide does not indicate that it poses a risk to the terrestrial compartment, or is there long-term exposure. Therefore it is not considered necessary to submit data to determine the effects of silicon dioxide on other terrestrial non-target organisms.	Document IIIA, Section 7.5.6
N/A	Mammals	N/A	N/A	N/A	N/A	N/A	N/A	The environmental risk assessment for silicon dioxide does not indicate that it poses a risk to the terrestrial environment. The toxicity profile of silicon dioxide as shown in Document IIIA, Section 6 Toxicological and Metabolic Studies does not indicate a concern regarding toxicity to mammals. It is for these reasons that it is not considered necessary to determine the effect of increased silicon dioxide exposure to mammals. Given the above justification, it is not necessary to submit data to meet the following data end points: 7.5.7.1.1 Acute oral toxicity (mammals) 7.5.7.1.2 Short term toxicity (mammals) 7.5.7.1.3 Effects on reproduction (mammals) Note that these points have been addressed for silicon dioxide in Document IIIA, Section 6 Toxicological and Metabolic Studies. Further studies are not required.	Document IIIA, Section 7.5.7.1.1 Document IIIA, Section 7.5.7.1.2 Document IIIA, Section 7.5.7.1.3

Footnotes

1. Due to the results available in the core base set of environmental toxicity data for silicon dioxide, particularly that available on the toxicity to earthworms and the fact that there is no exposure to the terrestrial environment, it is not necessary to submit further studies on the effects of silicon dioxide on the reproduction of earthworms or other soil non-target macro-organisms (the data requirements detailed in Document IIIA, Section 7.5.2.1).

2. Due to the results available in the core base set of environmental toxicity data for silicon dioxide, particularly that available on the toxicity to plants and the fact that there is no exposure to the terrestrial environment, it is not necessary to submit further studies on the long term effects of silicon dioxide on plants (the data requirements detailed in Document IIIA, Section 7.5.2.2).

4.2.4 Non compartment specific effects relevant to the food chain (secondary poisoning)

Result
The assessment of the potential impact of substances on top predators is based on the accumulation of hydrophobic chemicals through the food chains. Ideally a comparison between concentrations found in top predators should be made with the no effect concentration for that predator. As this data is not available a theoretical assessment is made. The first step in the assessment is to consider bioaccumulation potential. This has been assessed as unlikely to occur. Next the classification on the basis of mammalian toxicity is considered. Amorphous silicon dioxide is not classified as dangerous therefore it is not in the list of classifications to cause concern (i.e. R48, R60, R61, R62, R63, or R64.) In addition there is no indication of genotoxicity, although not directly relevant for the environment it may be indicative for top predators. Because amorphous silicon dioxide is not classified in the categories above and because there are no other indications, such as endocrine disruption, it is not necessary to perform an assessment of secondary poisoning. (Technical Guidance Document on Risk Assessment Part II Chapter 3 Section 3.8.3.1 (2003)).

5. HAZARD IDENTIFICATION FOR PHYSICO-CHEMICAL PROPERTIES

a. Thermal stability and identity of relevant breakdown products

Amorphous silicon dioxide is stable when stored at 54°C for 2 weeks. It is also well known that amorphous silicon dioxide is thermally stable (Melting point: ca. 1710°C).

For further details refer to Document IIIA3 Section 3.10.

b. Flammability and flash point

Silicon dioxide is a non-flammable solid that does not support combustion. Silicon dioxide does not exist as a liquid at normal atmospheric pressure therefore it is technically not feasible to determine its flash point.

For further details refer to Document IIIA3 Section 3.11 and 3.12.

c. Explosive properties

Silicon dioxide is thermodynamically stable, so does not exhibit explosive properties.

For further details refer to Document IIIA3 Section 3.15

d. Oxidising properties

Amorphous silicon dioxide does not readily release oxygen, is highly stable (melting point >1700°C, boiling point > 2200°C, low solubility ~100 mg/L) and only readily reacts with strong acids such as HF. Notwithstanding this, examination of the structural formula of amorphous silicon dioxide, along with the fact that it is widely accepted that amorphous silicon dioxide is thermodynamically stable, suggests that amorphous silicon dioxide will not exhibit oxidising properties.

For further details refer to Document IIIA3 Section 3.16

e. Reactivity towards container material

There is no specific packaging material which is known to be incompatible with amorphous silicon dioxide. Currently, amorphous silicon dioxide is packaged into 10 kg, 3-ply HDPE lined paper bags. Storage should be at normal temperature (around 10 to 50°C), and pressure. Amorphous silicon dioxide is stable on ageing at 54°C for two weeks as is its packaging.

For further details, refer to Document IIIA3 Section 3.17